How should science position itself with respect to issues faced by society? The persistence of the 'Ivory Tower', where scientists tend to hide behind their expert status to evade their responsibility towards society as a whole is intriguing. Especially for those of us who collect data in the field, societal issues cannot be ignored. Just two examples: as I was tracking mountain gorillas in Congo, I encountered a man-made track where snare traps were placed to capture whichever animal would be unlucky enough to place its foot or hand in the trap. Such snares were also used to trap gorillas, leading to very bad, even fatal, injuries. Could I close my eyes and let gorillas be trapped in them? Or should I remove the snares with the risk of entering a conflict with the snare owners and the habits of the local people? Later, in Taï forest, a poacher entered our research area and killed one of our habituated chimpanzees for meat. Should I refrain from intervening, because I was a researcher working in a foreign country?

So, what did you do? In both cases, I intervened. I removed the snares and had the poacher brought to court. By doing so, I became a party in the conservation landscape. I had to explain to the local population the reason for my reaction, and most villagers understood why I should act to protect our study subjects and the justifications for having the national park rules effectively enforced. Then the next question is whether we should only care about our study animals or whether we don't also have a responsibility towards all of the members of that species that face the same problem. As scientists working on them, we are the ones who know best what such threats can mean to the survival of the species, so, we are also the ones that could make the best case to protect them. For me, a logical consequence of all of this was, besides my studies, to create the Wild Chimpanzee Foundation (www.wildchimps.org), a non-government organisation working at the grass-roots level in West Africa to help conserve chimpanzees and their forested habitat.

What are your next projects? I am fascinated by what technological progress allows us to do. Ten years ago, the adaptation of genetic techniques to degraded DNA allowed us to undertake for the first time a genetic study of wild apes, determining paternity and reproductive success. Five years ago, we could implement for the first time projects about hormones in wild apes and uncover some of the effects of dominance and stress. Now, we are starting to measure the contribution of meat to the diet in chimpanzees and bonobos using stable isotope measurements. What will be possible tomorrow is not clear, but we will probably be able to measure things that cannot be measured in wild apes today. We have, therefore, launched the Pan African Chimpanzee project to collect data and samples from as many different chimpanzee populations as possible, before they go extinct due to human impact. This database will allow us to answer many questions about the factors promoting culture, hunting, tool use and other aspects in our closest living relatives. We may even uncover new facets about chimpanzees, whose future is so badly threatened. It is motivating to see how much more there is to learn.

What have the chimpanzees taught us? Humans have for as long as they could think and talk wondered about what makes us so special and distinguishes us from other animals. Here, chimpanzees, as our closest living relative, act as a direct testimony to our past. For the first time in history, we are in the fortunate situation that we can learn from observations on chimpanzees about our similarities and differences to them and we, therefore, are finally in a position to specify and define human nature. What is puzzling to my scientific eyes is that too often scientists seem to have a hard time to accept or consider what chimpanzees tell us about ourselves. Furthermore, we are still far from knowing the full extent of chimpanzee nature. I remember vividly the day a few years ago in Loango National Park in Gabon when I saw for the first time chimpanzees using tools to extract honey from deep underground. This was an ability chimpanzees had not been thought to have, and even I, having worked for 30 years with them, was not expecting to see this. How much more will chimpanzees teach us in the future?

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Quick guide

Monopolin

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What is monopolin and how did it get its name? Monopolin is a protein complex that organizes chromosomal architecture at the centromere and the ribosomal DNA (rDNA) repeats. Monopolin was identified in *Saccharomyces cerevisiae* as a core complex (Csm1 and Lrs4) and two accessory proteins (Mam1 and Hrr25) that is required for reductional division of chromosomes during meiosis I. Unlike mitosis, during meiosis I, sister chromatid pairs align at the metaphase plate with their homologous chromosome pair, and then each sister pair segregates together to opposite poles. Then, in meiosis II the sister chromatid pairs separate from each other as in mitosis. A key question has been why do sister chromatids segregate as a single unit during meiosis I. Because monopolin mutants attempt a mitotic-type division in meiosis I, it was proposed that monopolin acts to crosslink/clamp the microtubule binding sites on sister chromatids together during meiosis I so that they segregate like a single chromosome.

The idea that monopolin bundles together microtubule binding sites on kinetochores gained further support from studies on the function of monopolin in the fission yeast *Schizosaccharomyces pombe.* Unlike budding yeast, where each kinetochore binds a single microtubule ('point centromere'), in *S. pombe*, each kinetochore binds 2–4 microtubules. Although Mam1 is not conserved in *S. pombe*, Csm1 and Lrs4 (Pcs1 and Mde4 in *S. pombe*) are conserved. Monopolin mutants in *S. pombe* do not have defects in meiosis I chromosome segregation but do display frequent lagging chromosomes during meiosis II and mitosis that are caused by merotelic attachments. Merotelic attachments occur when microtubules from opposite spindle poles attach to the same kinetochore, creating a tug of war that causes the chromosome to lag behind the other chromosomes in anaphase and often mis-segregate. Thus, in *S. pombe* monopolin could act to clamp together microtubule-binding sites on each kinetochore to ensure that they all attach to microtubules from the same pole. It is unknown why monopolin is not required for mono-orientation of sister

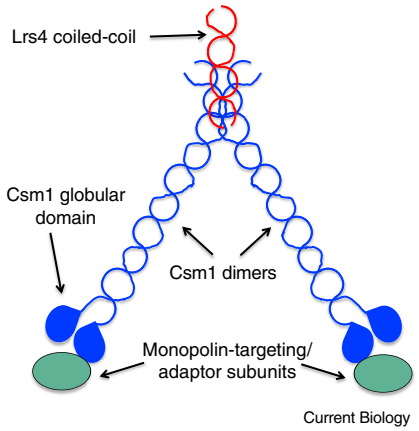

Figure 1. Structure of the core monopolin complex.
Monopolin forms a 'V-shaped' structure. Each arm of the 'V' consists of a coiled-coil dimer of Csm1 (blue) that ends in the Csm1 globular domain, which binds accessory/targeting proteins (green) such as Mam1. Lrs4 (red) forms a coiled-coil structure at the base of the V.

chromatids in meiosis I in *S. pombe*. One possibility is that the centromeric cohesin Rec8 takes the place of monopolin in promoting mono-orientation of sister chromatids in meiosis I in *S. pombe* and other organisms, consistent with the observation that *S. pombe rec8* mutants have defects in meiosis I similar to those of monopolin mutants in budding yeast.

Monopolin sounds a lot like the condensin and cohesin SMC protein complexes. Is monopolin an SMC family member? Based on primary sequence homology, monopolin does not look like an SMC (structural maintenance of chromosomes) protein. However, recent structural studies have revealed that at a gross level the monopolin complex has similarities to SMC complexes. As with SMC proteins, coiled-coil domains are a central structural feature of the monopolin complex. Overall, the core monopolin proteins Csm1 and Lrs4 form a 'V-shaped' complex (Figure 1). Each arm of the complex is formed by a dimer of Csm1, with an Lrs4 dimer binding to the base of the 'V'. At the ends of each arm the carboxyl terminus of each Csm1 molecule forms a globular domain that interacts with binding partners on the chromatin.

Does monopolin have other functions in the cell? The monopolin core complex (Lrs4–Csm1) localizes to the ribosomal repeats (rDNA) in the nucleolus and to the telomeres in *S. cerevisiae* where it promotes silencing and localization of these DNA regions to the periphery of the nucleus. As with any DNA repeat elements, rDNA repeats have the potential to become unstable through uneven recombination between repeat regions of sister chromatids. Because monopolin mutants have high rates of gain/loss of rDNA repeats, it was proposed that monopolin crosslinks rDNA repeat regions in sister chromatids to keep them in register and reduce unequal crossover events. Thus, as in meiosis I, monopolin could be acting at the rDNA repeats to crosslink chromosomal regions between sister chromatids. However, the functions of monopolin at the rDNA are likely more complex because monopolin also functions in rDNA silencing through the SIR proteins, and in positioning the rDNA at the nuclear periphery through interactions with inner nuclear membrane proteins.

Work from *S. pombe* has also shown that monopolin functions in anaphase spindle integrity. Monopolin localizes to anaphase spindles, and monopolin mutants display spindle breakage in anaphase. Given the function of monopolin at kinetochores, it is tempting to speculate that monopolin could act as a microtubule crosslinker to stabilize the spindle.

How is monopolin targeted to different regions of the cell? The globular domain of Csm1 is known to bind to distinct adaptor proteins to target it to different chromosomal regions. For example, during meiosis in budding yeast, the Mam1 protein targets Csm1 and Lrs4 to kinetochores, whereas other proteins target monopolin to kinetochores during mitosis and to the rDNA repeats. How monopolin is targeted to the telomeres and microtubules is not clear but likely involves additional targeting subunits.

Does monopolin always act as a crosslinker? Both the chromosomal segregation defects observed in monopolin mutants and recent structural data support the idea that monopolin acts as a crosslinking protein. However, other data suggest that monopolin could also function by recruiting other proteins. For example, in *S. pombe*, monopolin has an important role in recruiting the condensin complex to kinetochores. Like monopolin, condensin is required to prevent formation of merotelic lagging chromosomes. Interestingly, artificial targeting of condensin to kinetochores was able to significantly rescue the chromosome segregation defect in monopolin mutants, suggesting that a major function for monopolin may be in recruiting condensin. Similarly, monopolin was shown to recruit condensin to the rDNA in both *S. cerevisiae* and *S. pombe*. Further studies will be required to determine whether monopolin functions as a crosslinker or a scaffold for recruiting other proteins, or both.

Monopolin has been mostly studied in yeast. Does it function in other systems? Searches based on sequence homology to the budding yeast monopolin components Csm1 and Lrs4 only turn up obvious homologs in other fungi. Even among fungi, monopolin sequence homology between species is low. Thus, it is possible that monopolin exists in higher systems but has not been recognized because of poor conservation. Given the broad functions of monopolin in yeasts, it seems likely that functional homologs might exist in higher systems. Structural studies show that the globular domain of Csm1 is likely to be evolutionarily related to the Spc24/25 components of the NDC80 kinetochore complex, which is conserved between fungi and animals. Thus, it will be interesting to determine whether other structural homologs of Spc24/25 exist in animals or whether Spc24/25 carry out functions similar to monopolin in animal cells?

Where can I find out more?

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